

BRITISH HEART JOURNAL

Editorial

Doppler ultrasound and the apparently normal heart

Doppler ultrasound is such a sensitive means of demonstrating blood flow that it is now possible to detect flow patterns that previously went unrecognised and that might have been considered to be the result of a cardiac abnormality which warranted medical follow up, antibiotic prophylaxis against infective endocarditis, and even treatment. It now seems appropriate to attempt to distinguish between Doppler findings that should be regarded as normal variants and other findings which represent minor abnormalities. For practical clinical purposes, however, it may be more important to consider the management of minor abnormalities that cannot be detected clinically but are shown with Doppler ultrasound. Regurgitation through apparently normal valves is the most widely recognised of these abnormalities but others include small congenital defects and flow velocities just above quoted normal values.

Frequency of physiological regurgitation

Doppler ultrasound demonstrates regurgitation not detectable by clinical examination through valves that are normal on echocardiographic imaging. This was quickly recognised by Hatle and Angelsen¹ and subsequently has been widely studied. Such studies led to the acceptance of the concept of regurgitation through normal valves. The term "silent" or "physiological" has been coined for such regurgitation. In several precordial studies on healthy subjects of various ages differences in the frequency of Doppler detected regurgitation have been reported. Jobic *et al* summarise these studies in their article on page 109.² Pulmonary, tricuspid, and mitral regurgitation commonly can be demonstrated in apparently normal hearts in all age groups.^{3,4} Jobic *et al* found them in 100%, 100%, and 56% respectively.² The reported incidence of aortic regurgitation varies considerably. Some investigators never found it in children or young adults.⁴ It does occur, albeit rarely, in this age group and becomes a definite entity in older patients, with the incidence increasing progressively with age from forty years^{5,6} to a reported rate of 89% in those over eighty.⁵ It has also been suggested that regurgitation through all valves increases with age as part of a degenerative or "wear and tear" process.^{5,6} If this is the case it may not be terminologically correct to call the regurgitation "physiological", but rather "insignificant" and related to the changes occurring in the valve. The term "physiological", however, conveys the concept that such regurgitation is a common finding in healthy subjects, has no pathological significance, and should be regarded as normal. Continuing use of the term "physiological" should ensure that the finding is not regarded as abnormal.

Criteria

Studies of the incidence of "physiological regurgitation" have considered the criteria used to define its presence. It is generally agreed that the signal on colour Doppler echocardiography should be localised to the immediate area of the valve coaptation site and its duration should be between 100 and 200 ms on colour M mode⁴ or spectral Doppler echocardiography.⁶ Some would suggest that the velocity should be about that expected from the Bernoulli formula, but because a signal may not be present throughout the whole of systole or diastole the maximum velocity and pressure drop cannot always be measured. Sahn and Maciel consider that it is necessary to show acceleration of retrograde flow across the valve, if possible with aliasing at the regurgitant site—to suggest true regurgitation through a restrictive valve orifice rather than backflow as blood is pushed by the closing valve.⁷ These are essential considerations in scientific studies of physiological regurgitation—but what is their relevance to clinical cardiology? Physiological regurgitation can be diagnosed with some confidence when the regurgitant flow on spectral or colour Doppler is confined to the area very close to the valve, though it has been demonstrated by colour Doppler as far as 2.0 cm and even 3.0 cm from the valve.² As with most physiological variables, however, there is unlikely to be a clear cut-off point at which regurgitation can be considered to be physiological or pathological, and it will not always be possible to distinguish between physiological and mild pathological regurgitation. An attempt to do so brings in all the problems of trying to determine the severity of regurgitation with Doppler echocardiography—physiological variation with time, Doppler filter and gain settings, the colour algorithm used, and the fact that the extent of the colour jet is more dependent on the driving pressure than on its volume.⁸ In addition there are differences in the ability of different ultrasound machines to demonstrate regurgitation on spectral and colour Doppler echocardiography⁹ and the findings in one cardiology laboratory will not necessarily be comparable to those in another using different equipment and operators. With transoesophageal echocardiography it is likely that regurgitation will be detected more frequently.

What action should be taken when trivial regurgitation is found and it seems to be physiological? It is appropriate to record the finding on the ultrasound report lest its presence on a subsequent study be regarded as an alteration, but it is essential to ensure that the requesting physician is aware that this is a normal finding. Since this is a normal variant there is no need to inform the patient or his relatives; if it is raised in discussion it is important

firmly to state that the finding is normal. This should avoid the possibility of an imagined cardiac defect and prevent the generation of a population of people with iatrogenically created "heart disease".^{5,7}

Rheumatic fever

Left-sided valve regurgitation becomes clinically important in areas where rheumatic fever is prevalent. An apparently higher incidence of regurgitation has been shown in subjects suspected of having rheumatic fever but with no evidence of carditis.¹⁰ It has thus been suggested that the presence of regurgitation indicates carditis and that consideration should be given to adding left-sided regurgitation to the accepted Jones criteria for the diagnosis of rheumatic fever. Whereas aortic regurgitation in a young person is likely to be pathological the same cannot be said for mitral regurgitation unless it is clearly moderate or severe, and this should be apparent clinically. Doppler criteria for regurgitation must be applied carefully in rheumatic fever and their use by those who are unaware of the concept of physiological regurgitation could result in the overdiagnosis of rheumatic carditis and perhaps even rheumatic fever.

Implications

Doppler ultrasound shows flow through congenital defects that are small and minor, cannot be detected clinically, and would not have been diagnosed or investigated in the past. Such flow patterns can be demonstrated through a tiny ductus arteriosus,¹¹ through a ventricular or atrial septal defect, and through tiny vessels, probably branches from coronary arteries, which enter the pulmonary artery. Similarly it is now apparent that tiny leaks persist after apparently clinically successful surgical closure of a ductus arteriosus.¹² Received wisdom might indicate that such lesions, often with a high pressure drop and thus flow velocity, could be associated with the risk of infective endocarditis. And this could extend to physiological regurgitation. No one has suggested that antibiotic prophylaxis against infective endocarditis be given for physiological regurgitation, and I do not routinely recommend prophylaxis for a silent ductus or ventricular-septal defect. But is there a difference between physiological mitral regurgitation and trivial regurgitation with echocardiographic mitral valve prolapse? Is the risk negligible because the jet does not extend far into the receiving chamber and does not impinge on the distal wall? Large, complex, and long-term studies are required to answer these questions. Because infective endocarditis can occur in an apparently normal heart, however, such patients should have detailed Doppler studies to determine whether a minor abnormality is causally related to the infection. A corollary might be that because unrecognised abnormalities, such as these Doppler findings or a bicuspid aortic valve or mitral valve prolapse, may be present in apparently healthy individuals, all should receive antibiotic prophylaxis against infective endocarditis. But this calls into question the management of minor cardiac defects rather than the management of physiological regurgitation. If several healthy people have a tiny ductus arteriosus and none develops infective endocarditis related to this, is it really correct to undertake trans-catheter or operative closure of a tiny duct just because a murmur is audible? Similarly if residual flow is shown on Doppler echocardiography after a procedure, should further intervention be undertaken? The wisdom of intervention for a small ductus arteriosus has even been questioned.¹³ It has also been suggested that after the insertion of a ductal occlusion device a small leak without a continuous

murmur should be accepted and that there is no need to insert a second device,^{14,15} though antibiotic prophylaxis is probably prudent when prosthetic material is left in the great vessels. The management of relatively minor cardiac defects is further complicated by the Dutch report that concluded that the risk of endocarditis as the result of a medical or surgical procedure is small, and questioned the efficacy of and the need for routine antibiotic prophylaxis in the prevention of infective endocarditis.¹⁶ The answers are not known and long-term studies are needed.

Normal flow velocities in the ascending and descending aorta and pulmonary artery are less than 2 m/s.^{17,18} If there is a soft murmur and normal valve on imaging does a slightly higher velocity of about 2 m/s indicate mild stenosis, and is it thus appropriate to follow the patient every few years and recommend antibiotic prophylaxis against endocarditis? This carries the risk of heart disease being imagined by the "patient": there were similar problems with mitral prolapse in the past. It may well be that such echocardiographic findings do represent a mild abnormality but any problems, such as calcific aortic stenosis, will not become apparent for years and regular follow up will be unnecessary and inappropriate.

A B HOUSTON

Royal Hospital for Sick Children,
Yorkhill,
Glasgow G3 8SJ

- Hatle L, Angelsen B. Doppler ultrasound in cardiology. Physical principles and clinical application. Philadelphia: Lea and Febiger, 1982; 161-2.
- Jobic Y, Slama M, Tribouilloy C, Lan Cheong Wah L, Choquet D, Lesbre J-P. Doppler echocardiographic evaluation of valvular regurgitation in normal subjects. *Br Heart J* 1993;69:109-13.
- Kostucki W, Vandenbosche J, Friart A, Englert M. Pulsed Doppler regurgitant flow patterns of normal valves. *Am J Cardiol* 1986;58:309-13.
- Yoshida K, Yoshikawa J, Shakudo M, Akasaka T, Jyo Y, Takao S, Shiratori K, Koizumi K, Okumachi F, Kato H, Fukaya T. Color Doppler evaluation of valvular regurgitation in normals. *Circulation* 1988;78:840-7.
- Akasaka T, Yoshikawa J, Yoshida K, Okumachi F, Koizumi K, Shiratori K, Takao S, Shakudo M, Kato H. Age-related valvular regurgitation: a study by pulsed Doppler echocardiography. *Circulation* 1987;76:262-5.
- Choong CY, Abascal VM, Weyman J, Levine RA, Gentile F, Thomas JD, Weyman AE. Prevalence of valvular regurgitation by Doppler echocardiography in patients with structurally normal hearts by two-dimensional echocardiography. *Am Heart J* 1989;117:636-42.
- Sahn DJ, Maciel BC. Physiological valvular regurgitation. Doppler echocardiography and the potential for iatrogenic heart disease. *Circulation* 1988;78:1075-7.
- Simpson IA, Valdes-cruz LM, Sahn DJ, Murillo A, Tamura T, Chung KJ. Doppler color flow mapping of simulated in vitro jets: Evaluation of the effects of orifice size and haemodynamic variables. *J Am Coll Cardiol* 1989;13:1195-207.
- Comparative evaluation of imaging and Doppler ultrasound systems for examination of the heart. National Health Service in Scotland, Ultrasound Equipment Evaluation Project. Common Services Agency; 1991 May. Report No EEV/91/3.
- Folger GM, Hajar R, Robida A, Hajar HA. Occurrence of valvar heart disease in acute rheumatic fever without evident carditis: colour-flow Doppler identification. *Br Heart J* 1992;67:434-8.
- Houston AB, Gnanapragasam JP, Doig WB, Lim M, Coleman EN. Doppler ultrasound and the silent ductus arteriosus. *Br Heart J* 1991;65:148-51.
- Sorenson K, Kristensen B, Hanson OK. Frequency of occurrence of residual ductal flow after surgical ligation by color-flow mapping. *Am J Cardiol* 1991;67:653-4.
- Sturridge M. Doppler ultrasound and the silent ductus arteriosus. *Br Heart J* 1993;69:193.
- Hosking MCK, Benson LN, Musewe N, Dyck JD, Freedom RM. Transcatheter occlusion of the persistently patent ductus arteriosus: Forty-month follow up and prevalence of residual shunting. *Circulation* 1991;84:2313-7.
- Latson LA. Residual shunts after transcatheter closure of patent ductus arteriosus. A major concern or benign "techno-malady"? *Circulation* 1991;84:2591-3.
- Van der Meer JTM, Van Wijk W, Thompson J, Vandenbroucke JP, Valkenburg HA, Michel MF. Efficacy of antibiotic prophylaxis for prevention of native-valve endocarditis. *Lancet* 1992;339:135-9.
- Hatle L, Angelsen B. Doppler ultrasound in cardiology. Physical principles and clinical application. Philadelphia: Lea and Febiger, 1982:72.
- Wilson N, Goldberg SJ, Dickinson DF, Scott O. Normal intracardiac and great artery blood velocity measurements by pulsed Doppler echocardiography. *Br Heart J* 1985;53:451-8.